Drug-Induced Liver Failure Masquerading as Late-Onset Wilson Disease: A Case Report

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Abstract

It can be difficult to specify a cause of drug induced liver injury when not due to acetaminophen or other common substances.

This is especially true in today's broad market of homeopathic remedies.

This article provides a detailed case review of ALF likely due to the use of alkaline water that was originally thought to be due to WD.

Introduction

Acute liver failure (ALF) and fulminant hepatic failure (FHF) are defined as hepatocellular dysfunction and coagulopathy with no previous history of liver disease within 26 weeks (1,2).

Drug-induced liver injury (DILI) is the second most common cause of ALF and accounts for approximately 10% of all cases (3).

As the use of alternative medicines including herbal and dietary supplements increases, the incidence of DILI has also risen (4).

The presentation of liver disease due to Wilson disease (WD) can be highly variable from asymptomatic to ALF and easily confused with DILI (5).

Case Presentation

A 35-year-old male with no known past medical history presented to the emergency department (ED) with transient vision changes, balance difficulties, a sensation of "time jumping forward", and generalized weakness for the past 2 to 3 weeks.

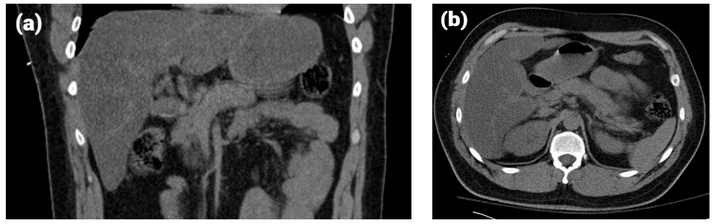
- On presentation his blood pressure was 84/42 with laboratory findings significant for acute renal failure, coagulopathy, and ALF (Table 1).
- CT scan of his head was unremarkable, and CT of his abdomen showed hepatomegaly and hepatic steatosis without evidence of cirrhosis (Figure 1). Abdominal ultrasound was negative for portal vein thrombus.

Due to reduced ceruloplasmin and the absence of other causes, the patient's presentation was thought to be due to Wilson disease (WD)

- A liver biopsy showed mixed portal inflammation suggestive of DILI and slit lamp exam was negative for Kayser Fleischer rings.

The patient's acute encephalopathy completely resolved after six days with supportive care only and he was able to be discharged home within 2 weeks with normalizing lab work.

As his mentation improved, he reported drinking alkaline water daily 10-days before the onset of his symptoms.



Variable (normal range)	Day of admission	5 days after admission	12 days after admission
BUN mg/dL (5-26)	18	102	38
Creatinine mg/dL (0.55-1.30)	2.11	11.11	2.0
Bilirubin, Total mg/dL (0.0-1.2)	2.1	5.0	1.3
Bilirubin, Direct mg/dL (<= 0.5)	1.6	2.9	
AST U/L (8-34)	11,867	193	65
ALT U/L (10-49)	1,001	171	289
Alk-P U/L (46-116)	152	135	126
INR (0.80-1.2)	5.37	1.19	1.0
Prothrombin time (9.3-12.4 seconds)	53	12.6	11.8
Ceruloplasmin mg/dL (16.0-31.0)	8.7	13.5	24.6
Copper ug/dL (72-166)	52	82	76
Urine Copper Level ug/L	2,787		
Urine Copper mcg/24hrs (9-71)			40
Alk-P (IU/L) to total bilirubin (mg/dL) ratio	72.38		

Table 1. Laboratory test results of the patient a day before admission, on the day of admission, five days after admission, ten days after admission, and two days after transfer to an outside facility.

Figure 1. Computed tomography images of the abdomen and pelvis. (a) Coronal view. (b) Axial view

Discussion

In WD, there is decreased secretion of copper into bile due to the absence or decreased expression of copper transporter in hepatocytes. This leads to increased hepatic copper accumulation, increased systemic circulation of copper, and decreased blood levels of ceruloplasmin.

- It is thought to be diagnostic for WD if serum ceruloplasmin level is < 18-20 mg/dL and if there is associated Kayser-Fleischer (KF) rings.
- Of note, ceruloplasmin is an acute phase reactant and can be decreased in the setting of acute severe end-stage liver disease or marked renal or enteric protein loss.
- A prospective study has found that the use of subnormal ceruloplasmin alone in the setting of ALF to screen for WD had a very low positive predictive value (5).

On account of rapid improvement in the patient's symptoms and liver function, the patient's ALF is now believed to be due to the consumption of alkaline water which is a previously reported cause when another specific offending agent is not identified (6).

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